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Karl Kastl
University of Nebraska Medical Center

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CUTANEOUS BURNS DUE TO DRY AND WET HEAT

SENIOR THESIS

1937

KARL KASTL

COLLEGE OF MEDICINE

UNIVERSITY OF NEBRASKA

OMAHA, NEBRASKA

480878

"The intelligence takes no cognizance of the vast calamity, I have seen, often deep and extensive burns, what seemed to be a living soul looking out of a crisp, charred and dead body. I have seen a child thus burned playing with toys an hour before death. Sometimes in this condition there appears to be a pleasant and mild delirium."

Wm. Hunt

OUTLINE

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Cutaneous Burns Due To Dry and Wet Heat

Introduction

In writing this thesis I will endeavor to give a general discussion of cutaneous burns which are due to dry and wet heat. On this topic itself one could write volumes and still not cover all ideas man has found in working in this field which is so important in modern medicine.

In reading Underhill (1) paper one finds that with the advance of industry, burns are becoming a greater frequency and consequently of greater importance to the practitioner. Their increase occurence lays an obligation on the medical profession to become conversant with the fundamental condition induced by a burn and to become familiar with most efficient methods of treatment. It is not sufficient to treat the wound only; much more important is the recognition of systemic effect and immediate institution of proper treatment. "What does a man profit if he dresses the wound beautifully and yet loses the patient."

A burned patient (1) should be treated in a manner similar to a patient with a crushed leg or

other extensive painful injury. Generally a surgeon does not operate immediately on a patient in shocked condition. He first attempts to reestablish circulatory efficiency and when the patient is in proper condition the operation is performed. In same manner the burned surface need not be treated until the circulation of the patient is again efficient. The patient does not die of injury; death is caused by circulatory failure of one or another type. Delay in treatment of the injured area will not be detrimental. Early infection need not be feared since there is little or no immediate absorption from the burned surface.

In J. H. Connell (2) discussion, he states that the Assyrians used bizarre salves to alleviate the pain and aid the healing of burned area. The modern Surgery text and editions not so long published a first aid treatment recommending carron oil. In any accident service a large number of burns entering have previously been treated with this inefficient, filthy, carron oil, butter, lard or other ingredients that only add to already difficult proper treatment. Most such first aid theropy is given by the well intentioned friend but too often also by the attending physician.

History

In burns the first thing man tried to do was to relieve the sufferer of the horrible pain, which is one of the major problems in present day treatment.

Hippocrates in 430 B.C. advised the following treatment (Sherman (3) "Having melted old swine's seam and mixed it with resin and bitumen, and having spread it on a piece of cloth and warmed it at a fire, apply with a bandage to the burned area."

A Greek, later, unknown, who lectured in Assyria, wrote a text on pathology and theropeutis recommends the following ingenious. "Roast a handfull of barley at the fire and mix it with oil of roses and white of eggs and use as an ointment."

The salve of Paracelus for wounds and burns is even more impossible. It consisted of very old wild hogs and bears heated half an hour in red wine, then dropped into cold water, which was next skinned and fat rubbed up with roasted angle worms and moss from the skull of a person hung, scraped off during increased of moon, to which where added bloodstone, the dried brain of the wild hog, red sandal wood and portion of genuine rummy.

Park and Davis (4) give a discussion, that at the time of Pare surgeons were prone to convert every wound and in incision into a burn, as it was their custom to sterilize the wound by cauterzing with boiling oil.

There, where many fallacious ideas in American villages which had its women charlaton, who continues to blow fire out of a burn. The technique involves certain incaulation and unintelligible mmmblings, accompanied by weird motion of the hands, in front of the blind-folded patient. The manoeuver is completed by blowing on the burn, after which the retained or imprisoned heat is supposed to leave.

Another fallacious idea still existent, is that fire or intense heat will remove pain from another burn, a sort of a homepathic principle of "Similia similibus curantur." This sees exemplification when burned individuals hold the injured parts to fire or source of heat in order to alleviate the pain. Shakespeare was cognizant of this when he wrote Romeo and Juliet. "One fire burns out another's burning." Our pain is lessened by another's anguish. (4)

(Park and Davis 4) The continuous water bath,

treatment of Helna, the open-air method and the closed or impermeable dressing for burns, the duodenal ulcer of Curling, and other features of burns and their treatment were discovered and described in literature many, many years before they enjoyed popular usage and came to bear the names of the men who supposedly were responsible for them.

"Every surgeon should understand that it is not he who heals, but the balsam within the body is that which heals, and that wherein thou art a good surgeon, is that thous offerest to nature defense and protection in the wounded part". "At this time, if at any time, the lady's hand is required", remarked Daniel Turner in reference to manipulation of burns: (Theophrastus 1493). The management of cutaneous burns had profited so little from medical progress that it led one surgeon to remark, "If the mummy of Ramises II should awake to reanimation he would find that the treatment of burns had slept with him during the long intervening centuries."

Maryland (5) stressed the importance of asepsis in treatment of burns in 1892. He gives the following statement:

"When a part has been burned whether by fire, water, metal, or other means, we may justly assume that the part has at the same time been sterilized; that is to say, heat has effected what we seemingly effect under other circumstances by use of our antiseptics." Treatment consisted at that time of the use of 1-2000 Bichloride of mercury in oil silk.

In burns (Wolfer J. (6) aseptic care is the curx of management. If infection can be prevented, the wound produced by thermal means will heal promptly with a minimum loss of tissue, beside that destroyed by heat. On the other hand when infection takes place, much tissue, which is partly devitalized and has the possibility of recovery, will become gangrenous and slough-away. The surface of a burn forms an ideal site for bacterial invasion; there is warmth, moisture and pabulum consisting of serum and devitalized tissue. In the more extensive burns much tissue may be destroyed, the slough again being the site of predelection for the invasion of bacteria, especially the anaerobes, such as gas-producing organisms. The majority of bad results obtained in treatment of burns are due to lack of aseptic care.

Despite considerable research (?) and undoubted advance in the methods of treating burns, the results at the present being obtained may still be criticised and improved upon. Perhaps too much has been expected of the tannic-acid treatment, which, despite modifications, is not in itself wholly satisfactory.

Prophylaxis and Statistic

Preventions of burns and scalds is of most important. (Park and Davis (4) 90% of deaths from burns is due to gross carelessness. Probably the best plan of procedure in eliminating the causes of burns is to educate the public. Hotels, ships, factories, hospitals and all public buildings should be fire proof and provided with sufficient permanent and utilizable exits, and external fire escapes as to permit a rapid evacuation at times of fire. The rigid inspection of the above institutions and the issuance of certificates of approval, if satisfactory, should be a part of the compulsory duties of the Public Health and Safety Dept.

It should be considered a legal offense to leave a child of tender years alone in a room with an open fire place, either of wood or gas or coal. If all burns were considered noticeable accidents, the police would inquire into each cause in order to properly fix the blame.

In New York City in 1924 there were 22,631 fires; as a result of these fires where 164 persons who died of these burns, 207 seriously injured and $19,000,000 worth of property destroyed. The establishment of a

fire prevention week in large cities is invaluable as a prophylactic measure against the loss of lives and property by fire.

Fire places should have protecting wire screens. The rekindling of fires by pouring oil on slumbering timbers should never be done. Use of oil lamps, their refilling and also of gasoline containers, cooking stoves, while the burners are lighted results in severe burns. Caustics and such inflammable substances as cleaning liquids, gasoline, ether, alcohol, and collodion should be handled with extreme care.

Children may be taught at an early age concerning the dangers of fire. They should be prohibited the use of matches. Hot irons, hot cooking utensils, hot soups, and beverages, exposed tubs containing hot water, should not be placed in reach of little children.

In factories, boiler explosions are guarded against by various safety precautions and devices; unprotected steam pipes are covered; electrical conductions are insulated and labelled with warning placards; acid vats are shielded. Aged people and epileptics are permitted to sit over fires, where they fall asleep or have a convulsion and are thereby burned. The windows are often thrown open, the draft from

which fans the flames to increased activity. The excited patient dashes into the open air and it is with difficulty that he is caught and the flames extinguished.

Pabst has devised a method of fireproofing clothing and drapery. One pound of ammonium phosphate in one gallon of cold water; the fabric to be treated is soaked in this colorless solution for five minutes, after which it is taken out and dried. The chemical is cheap and the solution is stable and uninjurious to the fabric which it impregnates.

In all instances of severe burns (4) prophylactic tetanius antitoxen should be routinely administered. The initial dossage, immediately given is 1,000 to 1,500 units: this can be repeated within a week.

The Eitiology and Incidence of Thermal Burns

(Park and Davis 4) "A burn is an injury inflected on the body by a degree of heat higher than is compatible with health action in the part effected." The types of thermal trauma are two: Burns that are produced by dry heat, and scalds by moist heat.

Wolfer (6) gives the following defination of a burn: "A burn is a wound produced by thermal means. It differs in no way from ordinary wound, unless, perhaps in extensive burns there is or may be considerable destruction of tissue which must be denuded before healing can take place. Burns may be so extensive so that it becomes necessary to consider loss of large amounts of an essential organ, the skin."

(Park and Davis 4) The skin is the most common seat of caloric injuries which are accordingly termed dermatitis calorica.

It is interesting to study the various physiological and pathological changes the body undergoes when exposed to high temperature of various degrees.

A leg or arm may be exposed for short time to excessive temperature 300 to 400°F in dry air, with impunity. A general exposure of the entire body to

to a temperature slightly higher than the normal inner temperature, is compensated for by an increase in the pulse and respiratory rate. Heat cannot be lost from the body in such temperature by radiation and conduction, but solely by evaporation, a process that tends to induce blood concentration.

Halliburton found all tissues are globulins coagulating at from 45-5℃ degrees. This coagulation of tissue protein is particularly dangerous when it involves the cardiac and respiratory muscles.

There is a varying degree of tissue susceptibility. Dense and thick skin (palms, soles, and buttocks) offer a greater resistance to heat than is offered by that of a thinner, softer, texture (neck abdomen, and axilla). All tissues are not equally susceptible to heat destruction.

The skin is more resistant than the mucous membrane and the latter more than the peritoneum. Of great importance is individual susceptibility. All individuals are not equally susceptible or sensitive to heat; thus it is a common experience that one person can handle hot objects that would burn another. The difference here is not entirely related to thickness of integument or sensitivity of the nerve endings for heat.

The progress of civilization and the improvement in the arts and science have greatly multiplied the frequency and severity of burns.

About 45% of deaths from burns occurs from birth through the fifth year. The frequency of burns and scalds in children can often be attributed to the careless ness of parents and servants and to the matured timerity and incautiousness of children. Severe and fatal scalds have been produced by placing children and infants in baths supposed to be properly tempered. Scalds frequently occur on small children falling into open tubs of hot water. Kettles containing boiling water, soup, or coffee are very frequently upset, spilling their contents on the upturned faces of little children.

Burns are more common and severe in instances where tactile sensation are subnormal or absent, as tabes dorsalis, acute alcoholism, coma of various types, epilepsy and paralyses. The injudicious use of hot water bottles applied to the bodies of anesthetized individuals for the purpose of inducing a better circulation occasionally results in a burn because pain "the warning sentinel of injury" is lacking. A paralyzed limb is burned or scaled at comparatively low

temperatures. Every surgeon has encountered burns of epileptics; patient and doctor alike are happily surprised to note a disappearance or lessened frequency of the epileptic attacks. This peculiar phenomenon was known by physicians of an early date, who herocially and unsuccessfully attempted to cure epilepsy by burning the patient.

Statistics for a long period of years show that 3 out of 5 deaths from burns are of women and girls. During the first three years of life, although they are similarly dressed, more boys than girls die of burns, probably because the ventursome spirit of the boys exposes him to greater danger. Figures of Metropolitan Life Insurance Company covering a period of over 20 years show that in the fourth year of life relatively high death rate of female begins. The margin increases from five to nine years when, more than twice as many girls as boys die of this cause, and there is more or less pronounced excess in the death rate for females until the age of thirty-five is reached. It is not until the age of thirty-five is reached where very large number of men are engaged in industries subjecting them to hazards of burns and scalds so many males die from this cause as do females. In later life when fewer men are subject to these risks,

women again experience a much higher death rate. Burns of women occur at home and arise usually from their household duties. Female attire is an important factor in increasing hazard.

The climatic influence are as following. Burns are more common in winter than in summer. Burns stand fourth in numerical importance among the causes of accidental death, being out-ranked only by automobiles, fatalites, falls, and drowning. Unfortunately, statistics concerning burns include death by conflagration, although all the deaths that occur in connection with burning buildings are by no means due to actual burns, but are often due to asphyxiation, crushing, etc.

Of all persons sick and unable to work in the various industries from 0.5% to 0.9% are incapacitated on account of burns. An approximate mortality rate is hard to estimate, because so many burned patients are ambulatory, but of hospital admissions, from 25% to 30% of patients die as a result of their injuries. The number of yearly deaths from fire alone, due to fireworks, varies between twelve to forty individuals; these burns are produced by giant fire-cracker and powder explosions.

Penberthy (8) gives statistics in his paper showing that 45% of burns which occur in United States occurs in children under six years of age. At Children Hospitals where 493 cases were studied the greatest number were between one and three years.

Bancroft and Rogers (9) stressed that all surgeons must appreciate the great economic loss of burned patients in the hospital. This loss is not only borne by the patient, but also by the public, for rarely does a ward patient pay his per capita expense to the hospital.

These figures show that 34% of cases were children under ten years of age. Deformites occuring at this age may result in their being public charges for many years. The memory of a burned child remains with the surgeon not only during the day, but often if he chances to lie awake at night, it might haunt him, particularly if he has a guilty feeling that at some time in the cause of the treatment he might have done more to improve the child's condition. Bancroft and Rogers feel that much must be done by public education both of mother and child to prevent this appalling percentage of burns in children.

Eitology of Burns

Eitological agent of burns vary according to (10).

1. The degree of temperature
2. The nature of the exciting agent
3. Its capacity for heat absorption
4. The duration of contact
5. The susceptibility of part acted upon
6. The condition of the patient

Dry heat of 140F and upward is capable of producing burns; scalds by moist heat of 125F and higher. The higher the temperature of the burning agent, the more severe injury with result, other condition being equal. Such burns are theoretically classed as scalds. But the lesions cannot be easily distinguished from burns caused by solid bodies.

Gun-powder explosions usually produce sever and deep burns.

Continued or prolonged exposure (4) of the skin to artificial heat produces an erythematous burn known as erythemus abigne. This appears as a moulding or marbled appearance of the skin, occurring for the most

part on old people, who toast their legs at the fire. It is also frequently observed on the chin and cheeks of cooks, stokers, steel mill and blast furnace employees (1). Fluids, such as oil, that boil at higher temperature than water produce increasingly severe results. The thicker the fluid is, at the same temperature, the greater is therefore its capacity for heat. Moreover, this oleoginous fluid adheres longer, and evaporation being slower, the effect is naturally more severe. As the heat of solid bodies is usually greaterthan that attained by liquids, except metals in a state of fusion, the former may produce very deep burns; white liquids flowing over a large surface, cause more extensive though comparatively superficol lesions.

Solids substances (4) as iron and steel, and the fixed oils (olive and linseed) cause more sever burns than aqueous materials. Fluids of the nature of alcohol and chloroform, produce only very superficial burns because of their volatility. The relative capacity of the substance for heat does not always determine the intensity of the injury, although in many instances this is true. The degree of inflammation is

proportional to the length of contact of the burning agent, a short period favoring a mild injury and a long period.

Metals in a state of fusion produce lesions that are usually of a greater severity on account of the high temperature to which they have been raised. (3)

Classification of Burns as to Surface area

The time-worn classification of burns into three or more "degrees" seems to serve no really useful function except as it tends to focus the attention of the surgeon in any given case upon the probability of the development of excess scar tissue formation (Culting 11).

"The classification of local superficial trauma into two degrees based on expectancy of scar tissue formation:

(1) Superficial

(2) Deep

as suggested by Bancroft and Rogers and others, seems more rational, and may eventually become the accepted nomeuclature. Under such a classification superficial burns would include all those which do not eventuate in complete destruction of the skin epithelium, especially such islands of epithelium representing hair follicles and coiled glands as lie at a lower level than stratum germinativum. Obviously, if a sufficient number of such islands remain undisturbed after a burn, they tend to act as natural Reverdin skin grafts from which relatively complete and normal epithelialization may proceed. The deep burns on the other hand would be

represented by lesions of greater penetration in which epitheliazation could not occur except from the edges of the lesions all islands of epitheluim having been destroyed, and which, consequently, invariably would require artificial skin grafting during the process or repair and possibly other surgical measures aimed at the production of satifactory functional and cosmetic results.

Aside from this very important practical difference between the lesions of burns and ordinary wounds, there is not very much of therapuetic novelty as far as burn lesions are concerned. Hemorrhage and infection tend to present problems in the one as in the other; especially is this true with respect to infection. The possibility of tetanus and gas bacillus infection should be carefully considered."

Underhill (12) discusses the importance of water in tissues. It is one of the most important nutritional requirements. The muscle and skin have a very important role in this, which is of importance in considering surface area of skin.

Dr S. G. Berkow (Park and Davis 4) has perfected a methods of estimating the extensiveness of burns and

scalds based on surface area. This is of value in determining surface area involved; which gives some idea of the prognosis, and its a very practiable method of classifying burns and scalds. It is more important for the physician to know the relative proportion of the body surface involved than it is to know the exact measurement of the dept. of burned area.

It is essential to know the relative proportion between the surface areas of the component members of the body and the total body surface. The whole is the sum of all its parts. The ratio between the size of lesion and the member of the body bearing it is easily judged. Berkow does his estimation as following:

"The ratio between parts and the total body surface arbitrarily corrected to the greater seriousness of lesions of the chest, abdomen, and genitals is,

Low extremites 38%

Trunk 38%

Upper extremities 18%

Head 6%

In children the proportions are different, the head and lower extremities vary considerably with age.

To ascertain the proportions at a given age the

the following rule is proposed:

Trunk 40%

Upper extremities 16%

for the head and lower extremities, subtract the age (in years) from twelve and add the remainder to the number expressing the adult proportion for the head (6%). Subtract the same amount from the number expressing the adult proportion (38%) for the lower extremities."

Berkow (13) estimates burned surface area in the following way:

"To estimate a lesion of the head, trunk, upper or lower extremities the number expressing the proportion of that part is multiplied by the fraction expressing the relation of the lesion to the part. For ease in arriving at the latter relation, it should be borne in mind that the hand is one fourth of an upper extremity; the arm, three fourths; that a foot is one sixth; a leg, two sixths; a thigh, three sixth of a lower extremity. The anterior surface of the trunk is 30%; the posterior surface 18%, the trunk includes the neck; the lower extremities includes the buttocks. If more than one part is injured, ~~the lesion of each part is~~

~~injured~~, the lesion of each part is estimated separately, and the sum represents the extensiveness of the total lesion. It is preferable to classify the extensiveness of lesion in each degree."

Extensive (14) superficial burns involving considerable areas of body surface is the greatest factor of importance in the development of the syndrome characteristic of burns.

Local Changes in Burns and Scalds.

Park (10) in considering local history, considers three stages:

1. The stage of destruction or burning
2. The stage of inflammation and sloughing
3. The stage of repair

Clincially it is very difficult to identify the "degree" of burns but for the purpose the local pathology I believe discussing what destruction occurs in different degree will give a much more clear-cut picture present.

Park and Davis (4) give the following description of local pathology:

"Degree one begins with a simple erythematous flush. The vascular reaction are similar to those of any inflammation, consisting of a mementory contraction, then a vasodilatation of the arterioles and venioles. This local widening of the capillary bed, due to the action of the irritant, is responsible for increased rapidity of blood flow to the injured part, causing the area to become warm and red. Immediately surrounding this injured area is a widely spreading irregular margin, exhibiting a bright arterial flush, which is

result of a local reflex causing a delatation of arterioles. While the stream bed remains wide, the current slows, and an active congestion of the part ensues.

There is a locally increased permeability of the wall of the minute blood vessels, so that a filtration of plasma occurs out into the tissues spaces, resulting in inflammatory edema. This superficial skin edema is responsible for the low flat wheals of various sizes that are found. Within the blood vessels, margination of leukocytes occurs with a subsequent migration into the tissues spaces, followed by a diapedesis of the red blood cells. The period of edema ordinarily lasts from thirty-six to forty eight hours. Within a few days the upper layers of the epidermis separates in the form of scales, or occasionally peels off. Any pigmentation which remains disappears in time. The linear fissures of the skin appear more prominent than usual because of the partial detachment or semidetachment of the intervening skin. The burned area may show an increased redness for a week or more. No scars remain. This type of burn is often due to the transient application of flame over

a great part of the body, as in gas explosions.

Second Degree: Degree two is one of vesication. The epedermal cells have undergone a true coagulation necrosis, due to the conversion of their soluble colloids into the insoluble "pectous" modification. An exudation of fluid passes from the tips of the papillae into the epidermal layers, where the cells which have been killed or injured by the heat, are swollen and soon dissolve completely. This passage of fluid has been attributed to the liberation in the skin of a diffusible substance having a histamine-like action on the minute vessels. It takes place at first immediately over the papillae, the interpapillary cells remaining intact for a while, until they are stretched, distorted, and finally dissolved by the increased volume of fluid exudate. The serous exudation is accompanied by an infiltration of mononuclear luekocytes. The rapidity of the exudation is dependent on the intensity of the stimulus, the sensitiveness and the vascularity of the part affected.

In mild cases the various sized vesicles or collections of fluid remain within the epidermis, leaving the basal cell layer or stratum germinativum attached

to the corium. In the more sever types, the fluid collects more rapidly and in greater amounts, beneath the epidermis, forming a bulla instead of a vesicle. This bulla lies between the epidermis and corium. MacLeod believes that the heat converts the moisture within the epidermis into steam, which separates the prickle cells layer, forming irregular spaces, facilitating the collection of serum. The volume of the vesicle varies from that of a pinhead up to that of an orange, ~~their dimensions~~.

Occasionally the surgeon will see a patient in whom the epidermis of an entire limb is elevated by a huge blister; A condition which must necessarily establish an enormous drainage upon the system. The extent and size of the bullae are dependent on their position in the skin, being larger in areas where the skin is thin and much less marked in proportion where the skin is thick. The thicker the epidermis, the more difficult is the production of blisters. Burning clothing, scalding fluids and the direct application of the flame are more likely to cause blisters than contact with a highly heated body. When the death of the skin has been subsequent to the injury, dark sanguineous vesication may form as in ordinary gangrene.

The contents of burn blisters are variable. Morner found 5.03 per cent of proteins which included 1.359% of globulin and 0.011% of fibrin. Engel states that the amount of protein in blisters fluids is proportional to the amount in the blood. Blister fluid may be either a transudate or an exudate, depending on the severity of the irritation. With a more intense stimulus, the capillary permeability is so altered that the fluid resembles plasma rather than serum. This plasma has a high and variable content of fibrinogen, which clots and converts the fluid into a fibrinous exudate. If this blister is not emptied of its contents, organization of the fibruious exudate is more likely to occur than spontaneous absorption. The clear serum within the blebs becomes cloudy in twenty-four hours. A substance reducing copper oxide is present in an amount equivalent to 100 mg of sugar per hundred cubic centimeters (Park and Davis 4). Blister fluid contains antibodies of all sorts, including amboceptors sufficient for complement-fixation test (Esenberg, Busckke and Zemmerman.)

Occasionally, if the vesicles are prevented from rupturing, the fluid content is absorbed, and epider-

matization proceeds beneath the unbroken cuticle. When the blister ruptures due either to an accident or a loss of elasticity with spontaneous breaking a continuous discharge of serum occurs on the burned surface. The roof of the vesicle becomes a thin, wrinkled pellicle which is movable over the reddened tender base. When the cuticle is removed, the cutis vera, red and painful, is left exposed. If the serum is drained off repair occurs quickly with covering of the corium by their new epithelium. Scaring will not result if the corium is not involved, and infection remains absent. The burned area may remain red and pigmented for a month.

Third Degree: In the third degree the epidermis is completely destroyed and part of the corium, but the tips of the interpaillary processes remain intact. This is the most painful type of burn because the exquisitely sensitive terminal nerve filaments are left bare and exposed. The papillae of the skin appear as a reticular framework, containing serum, bits of persistent living epithelium, leukocytes and masses of fibrin. The papillae of the injured portion are visible as red points on a white ground. The subpapillary plexus of blood vessels and lymphatics absorb the toxins formed in the burned skin. Occasion-

ally the sweat glands attempt to excrete these poisons, and a skin eruption occurs which simulates the rash of scarlet fever.

Tiny sudaminous vesicles may sometimes be found surrounding the burned area. The sebaceous and sweat glands and hair follicles are deep enough to remain uninjured, so that when the process of healing is inaugurated, each of these structures serve as a focus of potential epithelial growth, and the denuded area is quickly covered with new epithelium. The formation of this new skin requires from fourteen days to four weeks. The resulting scar is white, elastic, possessed of all the structural elements of true skin and undergoes no contraction.

Fourth Degree: Degree four involves destruction of entire integument. In every burn there are two tissue layers to be considered. The dead or destroyed tissue and beneath this, the injured or sick tissue. In this degree of burn the skin has been disorganized by the heat. The tissues are mortified. The dead skin forms an eschar, which is brownish or blackish and dry like leather, if produced by flame, or white, marble-like and coriaceous if burned by steam at high pressure.

The white or gray skin of the latter is due to the fact that blood cannot circulate through the vessels of the corium: consequently, the color changes normally produced by finger pressure will be absent.

The eschar is insensible to touch. The eschar is depressed below the level of the surrounding skin, which is drawn in around it, showing puckered folds and corrugations radiating from the periphery of the disorganized area. The area about the eschar gradually shades off into hyperemic zones in which the burns are of the third, second and first degrees. In a short time an acute inflammatory process starts around the retracting eschar, and a groove results, intervening between the edges of the dead and living tissues. This is the initial step in sloughing, a process that ordinarily requires two weeks for completion, providing it is not hastened by intervention.

The eschar occasionally is cracked or fissued, especially near the joints, where movement occurs, rupturing the dry and brittle skin. At times these fissures appear immediately after the injury, and the split skin extends down to the subcutaneous tissue. Here the fat cells of the panniculus adiposus lose

their oily contents, due to the melting by the heat, and the released fat flows out over the edge of the fissure on the surrounding skin. If any part of the corium remains, some of this fat may be disseminated throughout it in the form of small granular masses. It is astonishing that arteries and nerves sometimes preserve their vitality for several days in the midst of this disorganization. However, blood contained in the vessels of the burned area of the fourth, fifth, or sixth degree is coagulated, and the precise depth of the burn can be determined by the level at which bleeding occurs on incisions. The process of healing begins soon after the injury, but is not so evident until sloughing or mechanical removal of the dead skin occurs. The remaining debris is cleaned up or liquified, partly by autolysis, partly by leukocytic digestion, the residue either flushed off the surface of the wound or absorbed via lymphatics. In severe scalds of the hands and feet the nails become detached. Infection and suppuration only too frequently accompany the separation of the nurotic tissue.

The raw surface is covered with a fibrinous exudate, which exerts a chemotactic or thigmotopic in-

fluence on the growth of new tissue cells. The exposed ends of the blood vessels are closed by little plugs of thrombi, but from these capillaries, tufts of endothelial cells, accompanied by fibroblasts, grow out along the fibrinous framework and organize it into new tissue, known as granulation tissue. The sprouts of endothelial cells hallow out into tubes forming new anastomosing capillaries, which arch and thereby give the granular appearance to the surface of new tissue.

The granulation tissue is in time covered by a thin bluish film of epithelium, which grows in from the periphery at the rate of one eighth of an inch a week. The new epidermis later becomes thicker and opaque. A good deal of contraction occurs, and scarring is inevitable. The scar by its cicatricial contraction is a common cause of deformity. The granulation tissue has a marked tendency to become over abundant and luxurious, a condition which hinders epithelialization, and makes the resultant scar irregular, inelastic, protuberant and contracted. The scar may be smooth and shiny due to the absence of such epidermal accessories as hair follicles and sweat glands. The

border of the sac is irregular, indental and occasionally stellate.

Fifth Degree: In degree five the muscles are encroached on. There is no essential difference from a burn of the fourth, except that the surface is more deeply charred. The scar is deeper, firmer and immobile. There is greater disfigurement, depending on the importance of the muscle involved, considerable functional impariment may result the scar has a decided tendency to break down and ulcerate.

Sixth Degree: In degree six the tissues are charred and carbonized, being converted by the heat into animal charcoal. The fingers and toes are the parts most fequently exhibiting this degree of destruction. A larger number is seldom the seat of carbonization, unless the patient was insensitive, paralyzed or forcibly prevented from movement at the time of injury. The heat may fracture the bone.

General Tissue Changes in Burns and Scalds

(Park and Davis 4) give the following review:

"There are no internal or visceral lesions pathognomonic of burns and scalds. Cumin first reported autopsis on burned patients in 1823. As early as 1840 Long noted that the pathologic changes in the internal organs closely resembled those due to acute febrile diseases involving the skin. The toxic for parechymal cells.

Many investigators have attributed a goodly portion of the visceral pathology to minute capillary thrombi. These tiny thrombi are assumed to be distributed ubiquitously throughout the body and by their plugging action, which occurs during life, produce a stasis and hyperemia in the lungs, kidneys, gastrointestinal tract, brain, liver, etc.

Brown-Sequard believed that the various remote effects produced by burns were brought about through the medium of the spinal cord, which he regarded as reflecting the irritation from the burnt part to the secondarily affected organs.

When a burn undergoes an exhaustive suppurating process, the amyloid infiltration of viscera, which

so commonly follows chronic destructive infections, is likely to ensue.

The normal (14) healthy, unbroken skin is composed of cellular or epidermal layer, the corium or connective tissue layer, in which are embeded some hair follicles and sweat glands, and the main vascular or capillary bed. The delicate capillary walls form a closed system. Each individual cells of the tissue constitutes a unit itself, receiving its food and oxygen and giving up its waste product through a membrane by means of osmosis and diffusion.

In burns these structures are subject to great heat, which may destroy the epidermal cell layer and leave the corium, through which pass the majority of the sweat glands and hair follules, to end in subcutaneous fat (Barnes 15). In a second degree burn, the physical continuity of the corium is unaltered, but its vital continuity changed. The outer layer of cells is destroyed, the deeper cells are so injured that the cellular unity is lost, and the intracellular contents pour out. The capillary walls are broken down, and each tiny vessel stands open to receive into its luman any substance with which it comes in contact,

~~with~~. The blood plasma contains many enzymes, and ferments of which is little known, pours out through the opening capillaries and mingles with the liberated intracellular contents containing proteases and other digestants essential to cell activity forming a fluid medium in which chemical changes begin at once.

Park and Davis (4) found the following necropsy results.

The Nervous System: Necropsy reveals hyperemia of the brain and meninges. There may be an effusion of blood between the dura mater and the bone. The white substance displays puncta vasculosa. The arachnoid vessels may be engorged with blood and contain occasional thrombi. Korolenko states that the sympathetic nervous system is seriously involved. The cerebral cortical cells and gangleon cells are uninjured except in instances of shock, in which the nerve exhaustion is accompanied by chromotolysis or partial destruction of Nissl's granules.(Crile).

Lungs: Within fifteen minutes after the occurence of the injury, an eosenophilia appears in the lungs, but this is not specific because it is found subsequent to all destruction of animal tissue, resulting in endogenous intoxication (Kotzareff). The lungs are

congested and may contain fibrinous plugs. Thrombi have been reported in the small branches of the pulmonary artery, obstructing the circulation and exerting additional strain on the right ventricle.

Kidney: The tissue toxin, being secreated by the kidney, produce an acute glomerulitis. Cloudy swelling and fatty degeneration later appear in the proximal convoluted tubules. The degeneration is accompanied by venious stasis which induces further tissue breakdown. Wertheim found thrombi in the kidney occuring most abundantly in the capillary tufts of the glomeruli with severe burns, necrotic foci become abundant in the kidney and grow larger and more extensive as the toxemia persists. These necrotic areas incite cell multiplication, and a proliferative process results, with irreparable damage to the kidney. Hemoglobin pigment is found in the kidney, being most abundant in the straight uriniferous kidney, tubules, although occuring also within Bowman's capsule and the convoluted tubules. This blood pigment is responsible for the dark brownish red color of the kidneys, as described in necropsy reports, and which has erroneously been attributed to excessive hyperemia. The hemoglobin is excreted through the glomeruli and appears in the

urine.

Suprarenal Glands: H. C. Weiskotten has made an intensive study of the pathology of the suprarenal gland in extensive bufns. Whereas the normal weight of the suprarenal gland is from 4 to 7 Gm, in burned patients its weight is often from 20 to 25 Gm. The peri suprarenal fat tissue is markedly edematous. The suprarenal gland of the young patient is more labile with reference to the burn toxin than is the adult gland. When the animals are killed 24 hours after burning, there are few changes in the cortex of the adrenal except hyperemia and occasional hemorrhages. The glands are swollen and deep red, due to hyperemia and ecchymotic areas of hemorrhage among the parenchymal cells. These pathologic changes are more or less in direct proportion to the extent of the burned lesion. The gland cells are swollen, pale staining, hydropic and frequently necrotic. The type of changes in the suprarenal is similar to that occuring in dephtheria intoxication and antiphylactic and peptone shock. In analysis, the epenephrine content is low or totally absent.

Heart: Necropsy occasionally reveals sub-endocardial and subepicardial hemorrhage. If the

toxemia has persisted for a sufficient length of time, the cardiac musculature exhibits areas of hyaline and fatty degeneration and necrosis of the muscle fibers. It is possible for the right ventricle to be dilated.

Spleen: The spleen is softened and enlarged. Focal necroses occurs in the germinal centers of the lymph nodules. The lymphoblasts undergo karyorrhexis and karyolysis and are rapidly ingested by phagocytic endothelial leukocytes. The endothelial leukocytes may fuse to form foreign body giant cells. These lesions reach the height of their development within seventy-two hours after injury. Later the lymphoid nodule appears homogeneous, due to hyaline degenerative changes.

Lymph Glands: Bardeen has described certain lesions occurring in all the lymphoid structures throughout the body, lymph glands, spleen, intestinal follicles, etc. (16). The germinal center of the follicle is first edematous. Swelling and distortion of the lymph cells of these areas are soon followed by necrosis and dissolution, the changes gradually extending peripherally. The clearance of this lymphocytic debris from the center of the follicle discloses large, flat endothelial cells containing faintly stained nuclic. Mc Crae states that

the proliferation of these endothelial cells is identical with that seen in typhoid fever and other acute infections. Bardeen (17) believes that toxin exerts its initial deleterious action on the center of the follicle because of the vascular arrangement of this follicle. This arrangement is such that a tiny arteriole runs to the center of this area and here breaks up into capillaries which radiate out from the center and are collected into veins at the periphery of the follicle. These anatomic studies were the work of Calvert.

Bone-Marrow: Necropsies on human subjects, and on dogs, experimentally burned, reveal focal necroses of the bone marrow, if the subjects live long enough to permit the start of an active leukopoeises. Burn toxin in mild amounts is positively chemotactic for leukocytes and stimulates the bone marrow to increased productive activity, but like many other irritants, an excessive quantity destroys instead of stimulates.

Gastro-Intestinal tract: The primary lesions of the gastro intestinal tract commonly caused by caustics will not be considered here, but only the pathology secondary to burns and scalds of the skin. The swelling and pathology changes of the solitary and agminated

lymph nodules are similar to the lesion described by Bardeen in other lymphoid structures. There is a generalized hyperemia of the mucous membrane, with punctate areas of petechial hemorrhage into the mucous membrane of the stomach and intestine, particularly in the ileum near the cecum. The ecchymosis may be large and at times produce an ulceration.

Serious Cavities: The pleural, peritoneal and joint cavities are prone to contain an accumulation of serious exudate, especailly when the burn or scald is of the skin overlying the serious cavity. The exudate is occasionally hemorrhagic.

Bardeen (16) gives the following findings: The morbid anatomy chiefly consists of cloudy swelling of the liver and kidney, softening and enlargement of the spleen; ~~Alone all~~ swelling of the lymphatic glands and gastro intestinal lymph follucles; These also are hyperemia of the thoraic and abdominal organs; These where the findings within 48 hours after the burn.

Curling ulcer of burns. Parks (10) gives a discussion on duodenal ulcer of burns. He found that duodenal ulcer is not a constant sequelae of burns and are admittedly uncommon, yet they do appear frequently, which would indicate they are not merely coincidental.

It is almost impossible to diagnose these lesions while the patient is living. Death occurring three to four weeks after burns seldom reveals an ulcer. The casual relation of the burns to duodenal ulcers is still held doubtful by some pathologists.

Curling believes that duodenal localization of ulcer is specific, but literature reveals many instances in which the stomach is also involved.

The mechanism of production of the ulcers is a mystery. It seems possible that the absorption of toxin may be the cause. (Novak 18).

Again some believe it is a vasomotor nerve reflex from the skin because of frequency of these ulcers in abdominal burns.

Novak (18) reports that ulcreation of the gastrointestinal tract following external burns has attracted attention ever since 1842. That year Curling reported 12 cases of external burns with subsequent lesions of the intestine. At an autopsy of these cases the lesions found were in the duodenum within an inch of the pylous.

Body Fluid Changes Resulting From Burns

There are many theories as to the possible causes of symptoms in sever burns. I will try and give the viewpoint of few of the different findings as related to pathology. Few of the theories are as follows:

1. Surface Dehydration and heat radiation disturbance.
2. Absorption of toxin
3. Decrease of blood volume.
4. Vasomotor disturbance.

Surface dehydration and heat radiation disturbance:

It is believed that shock in burns was due to dehydration through the burned area. Mc Clure (19) in a series of experements on rate of evaporation in 13 cases of first degree burns was done and the following was found. Where the rate of evaporation was calculated for the whole body over twenty-four hours period, it was ascertained that no significant changes in the weight of the body and no important losses in the total amount of fluid would be expected to result from evaporation alone.

The deleterious effects resulting from varnishing the total skin area of animals or absorption of atlered

metabolites, but to an increased heat loss and hypothermia Steward (20) cites an instance where a patient with cutaneous disease was treated by covering his entire body with tar and kept covered for ten days, without any noticeable interference with normal functions.

The excretion (4) of waste materials is one of the least important of the skin's many functions. The noxious retention theory of toxemia of burns is no longer tenable.

Lee (Steward 20) found that normal skin is a most efficient insulator and heat regulator of the body. The destruction of large areas of skin results in a marked loss of body heat through radiation with an increase in shock pending an application of proper treatment.

The advantage of keeping the burned patient in a very warm room or near a fire was recognized at an early date, but the reason for it only recently became generally known and accepted, largely though studies made during the World War.

Welti (Davidson 21) disproved the theory of failure of heat regulating mechanism by showing that animal die in spite of adequate protection against such heat dissipation.

Absorption of toxin. As early as 1868 Wenthen believed death in burns was due to a toxin produced

within the burn area (22).

The skin is composed largely of protein material which is broken down by heat, and this is subject to the action of various chemicals present (14). The whole area becomes a seething layer of brochemical activity, truly a stewpot of the "Grim Reaper", the brew from which ~~must as~~ is absorbed directly into the blood stream of the unfortunate victim of a burn. Each systole of the patient's heart adds fuel to the flame by pumping fresh plasma into the mixture, and each diastole sucks away from it the highly toxic products newly formed.

Clinical experience and experemental research have shown the toxic product poured so rapidly into the blood stream to be frought with due possibilities. Many different substances have been found, but no specific toxin has been isolated (14.)

The majority of investigators agree the chief offender is some product of protein metabolism which is manufactured at the site of the burn (21). Its manufacture and absorption begin at the inception of the burn, and enough to produce death may be absorbed within the first twelve hours.

The absorption (15) of toxic products occur mainly within the first 24 hours, by which time the reparative processes close the open capillaries, and any absorption thereafter is by osmosis through the reestablished walls of the capillary bed. Robertson and Boyed (22) have showed that a lethal amount can easily be absorbed within the first eight hours; hence, the primary treatment is of utmost importance in preventing subsequent toxin shock.

Toxemia of serious cases is practically always prevent, as shown by symptoms and necropsy finding, the fever of uninfected burns is no doubt due to the action of the released toxin on the medullary heat center. Is the toxin formed from injured blood elements or is it a product of the burned tissues? Scholz is of the opinion that the blood itself rather than the tissues is the seat of chemical change. (4). Ravenna and Misossian asserts that blood heated in vitio to 55 or 60° C is toxic and on injection produces identical visceral lesions with the burn toxin, whatever if may be. Pawlowsky denies the presence of a toxin in blood. The presence of the toxin in the blood does not necessarily indicate its formation there

any more than its detection in the urine establishes this fluid as the toxicogenic substance.

The present consensus of opinion favors the burned tissue as the source of the toxin, which is absorbed and circulation in the blood being carried by the red blood corpuscle (23).

Vogt (4) demonstrated the foregoing experiment on animals. If the burned area of equivalent size and severity were permitted to remain for eight hours, the animals invariably died: but the transplantation of this burned skin to a normal animal resulted in the latter animal becoming toxic within an hour, while the burned animal was saved from a toxic death by the transplantation.

Confirmatory evidence has been given by parabiosis when one of these animals is burned the other becomes intoxicated also, and the burned animals suffers less than would ordinarily be the case. If the united animals are separated within twelve hours after one is burned, the unburned animal does not develop symptoms. Heyde and Sauerbruch verified these parabiotic experiments. Hyde utilized this principle in disproving the theory of reflex action.

Salvioli, Markusfeld, and Steinhouse (4) found that after cutting of the blood supply to a rabbit's

ear, a burn of this ear would evoke no constitutional disturbance; but if the nerves to the ear are several and the blood supply left intact, toxemia results from the above experiements one very eaily sees that the poisonious substance is generated or elaborated in the injured tissue and cannot cause a generalized toxemia until it has been absorbed and circulated by the blood. Perhaps this is one explanation why an extensive superficial burn is more dangerous than a small deep one.

The symptoms of toxemia do not appear until after the first twenty-four hours, a fact which indicates, according to Robertson and Boyd, that the damaged tissue must be in contact with living tissue for some time before a toxic substance ~~with living tissue for some time before toxic substance~~ can be elaborated.

The toxin are eleminated by the kidney and intestines in which they produce lesions while in transit. The kidneys excrete these poison in quantities sufficient to make the urine of increased toxicity to rabbits. The urine of burned patients and animals contain an increased nonspecific proteolytic ferment, which is capable of splitting glycotryptophane.

It is stated that the very complexity and multi-

plicity of the theories adumbrated and volumes for our ignorance of the true nature of the burn toxin, and stamp this evidence as nugatory. The toxin of burns is unknown.

Best and Mc Henry (3) found the following result:

Extensive burns in human subjects and in animals may be followed by a state of shock which has certain points in common with the condition produced in animals by large doses of histamine. The concentration of the blood and the changes in blood chemistry are in many respects similar. An increased secretion of epinephrine and a decreased amount in the adrenal glands of cats have been reported to be produce by superficial burns. Although Robertson and Boyed (1923) extracted from burned skin substance which produce a shock like condition in normal animals, there is as yet no evidence that these toxic substances are absorbed or that they possess more than a superficial resemblance to histamine. One is not in a position to decide on the relative merits of the theories.

Davidson (21) supports the theory that toxin is produced in the burned area. He gives a case report where a minor burn was treated with tannic-acid in an

efforts to remove the coagulated tissue, there was a prompt and marked rise of temperature. In another instant which was case No 2 boric acid was used after precipitating the devitalized skin was immediately followed by a delirium and pronounced rise of non-protein nitrogen of the blood.

There are many who believe there is very little if any toxin absorbed from the surface area. As they have shown by their experiments. They seem to disaprove the toxic theory, but one has to consider both possibilities.

Underhill (1) performed the following interesting experiment. He took massive doses of strychinine that would kill a normal animal in a few minutes and injected it under and into the burned surface area causing no noticeable influence in the animal.

From a series of experiments by Wilson and Stewart (24) as to the depression action of Extracts of Burned skin resulted in the following summary.

(a) Trichloracetic acid extracts of normal skin of rabbits have a depressor activity which is not due to acetylcholine, aderosine, histamine, or the "P" substance.

(b) Extracts of rabbit skin to which heat had been briefly applied at intervals from three minutes to 48 hours previously, contain an apparently identical depressor substance. Such extract show no increased depressor activity, but sometimes a diminution, which is probably due to dilution of the depressor contant of normal skin by edemia fluid.

DECREASE OF BLOOD VOLUME IN BURNS AND SCALDS

(Underhill, Carrington, Kapsinow, Pack 14). Blood volume loss results in a disturbance of normal physiology of the cardiovascular system. Marked concentration of blood means a failing circulation, an inefficient oxygen carrier, oxygen starvation of tissues, fall of temperature and finally suspension of vital activities.

Underhill (12) found that the fluid lost at the site of the burn is the whole plasma of the blood. Until the capillory walls have been repaired there is a steady depletion of the blood plasma, which Underhill and his co-workers ~~and~~ Secondly ~~in~~ extreme dehydration due to the moblization of all available water in the tissues.

It is the increased viscosity of the blood which soon offers so great a resistance to contraction of the heart muscle, that the shock is either produced or increased.

In Harkins (25) paper he states that in traumatic shock and severe burns there is a marked shift of fluid from the blood stream into the injured vessels.

(Bloclock 26) fluid escaping into the tissue due to damage of the tissue was found to be of the composition as plasma of blood. After the blood volume is reduced it is likely that even small amount of toxin in the blood produces deleterious effects upon internal organs as discussed in general pathology of this thesis.

Studys of fluid present (Beard & Balalock 27) in injured area is shown to be greatly increased, and it is believed that the loss of plasma protein there is the most important factor in producing shock by experimental methods used on animals.

Weriner and Elman (28) believe that large amounts of blood plasma are lost into burned areas, has long been known. Its measurement experimentally, has shown that it may be very extensive. It is also generally realized that it is the prime, if not the only factor in the blood concentration of burned patients. But its significance in theropy has not been sufficiently emphasized.

In a number of several burned patients intravenous acacia or whole blood plasma was given. Under influence of this treatment the concentration of

erothrocytes was rapidly reduced and subcutaneous edema relieved: In several instances the red cell count dropped rapidly within 4 to 8 hours after injection of 500 to 1000 CC of acacia or plasma. In one patient the count fell from 8,200,000 to 6,000,000 within four hours after the use of 500 CC of 6% acacia in glucose plus 500 CC of physiological saline. Such a prompt relief of concentration was never observed following the use of saline or glucose alone.

In treatment of shock (29) the need is for a fluid that will stay in the vessels, restore blood volume, and maintain it until the normal fluid, regulating mechanism is again able to function.

Blood is the ideal fluid to use because it raises blood pressure, sustain it, combats anemia, which may bring about impairment of nutrition of the vital centers, and preserve colloidal properties. However, tranfusion of blood is not always possible and inadequacy of Ringer's Lock's, and physiologic salt solution has been generally admitted.

Balyiss (29) in 1916 found a solution of gum acacia ~~were~~ was efficacious in restoring blood volume.

These observations (Werimer and Elman 28) in-

dicate that loss of serum protein is a serious result of extensive burns, and that the store of body protein is not sufficient to restore rapidly this loss when only water, glucose, and electrolytes are administered. In severe burns, therefore, large amounts of protein are needed; The injection of blood plasma is apparently more efficacious than blood because of the excessive concentration of red blood cells already present. In addition to its direct effect in correction of lost protein, it is probable that the injection of blood plasma aid the body in its resistance to infection, which is an ever-present danger in serious burns.

The administration of too large amount of water, electrolyte and glucose alone as ordinarily carried out seems not only ineffective, but may, if excessive, lead to deleterious results by producing generalized edemia thus lowering tissue resistance.

Therefore, blood plasma or acacia should always be used. Keith (Lilly 29) states that the mode of action of acacia in restoring fluid to the circulation is not clearly understood. It is hoped that new knowledge about colloids may assist in the solution of the problem. Acacia leaves the blood stream in about

six days. The rate of excretion varies in different individuals. Good and Boyer have emphasized the value of acacia as a safe, ready, and inexpensive fluid use as an aid in increasing blood volume.

Camp (30) found that fluid loss varies proportionally with the extent of the burn in severe cases comprising as much as 70% of total body fluid. Red cells do not pass into this transudate and hemoglobin estimation to measure the degree of concentration in some cases shows over 200%. A reading of 125% indicates a precarious condition and 140% over an extended period of time is incompatible with life. The Chlorid shift is decreased, the greater blood volume loss, the greater is the chlorid loss.

The early use of glucose is not indicated according to Greewald and Eleasberd (Park & Davis 4) who observed, Hyperglycemia in humans and rabbits during the initial period of shock and attributed this blood sugar to hyperactivity of the suprarenal gland. When toxemia ensued, with the accompanying degenerative changes in the adrenal glands, they observed the hyperglycemia to change to hypoglycemia.

Urine Findings

Park and Davis (4) noticed change in composition of urine. In severe burns resulted in Oliguria because of two factors, kidney lesion impairing the renal secreation, and a concentrated blood with insufficient plasma to exert a hydremic, stimulus on any kidney. The urine is highly colored and often smoky because of the hemoglobinuria. The specific gravity is naturally increased. Albumen appears very soon after the reception of severe and even of almost immediately fatal burns.

Davidson (2) found in burns of mild degree only a faint trace of albumen is present on the first day, but it increases in amount on subsequent days. With each exacerbation of fever, albumen again appears. Acetonuria frequently appears by the third day after the burn. The increase in the ratio of the animonia nitrogen to the total nitrogen in the urine is probably due to ketoses. Various investigators have written of the presence of a protein of unidentified character in the urine of the patient after severe burns. Wilms noted an albumosuria as a common occurrence.

The Symptoms and Diagnosis of Burns and Scalds

There are three symptomatic stages in the history of severe burns or scalds (Park and Davis 4).

1. The period of irritation and depression. This occupies the first 24 to 48 hours.
2. The period of reaction and inflammation. This extends from the second day to the second week.
3. The period of exhaustion and suppuration. This extends from the second week to complete convalescense.

Mild and minor burns are often asymptomatic, except for local tenderness and pain. Hence, the discussion is limited to those burns and scalds capable of producing systemic or general symptoms.

During and immediately after the accident the patient is hyperexcited, and suffers most excruciating pain and agony. Proper treatment causes a marked abatement of this most distressing symptom. Within five or six hours the mental clarity may give away to apathy, punctuated by deep yawns and sighs, shivering and complaint of chilliness, together with deep inspiration hiccough and then vomiting. The shock which now super-

venes is nervous in character, and especially in fatal cases in preceded by restlessness, and at times convulsions. The last especially is common in children. Vomiting and convulsions are ominous.

When large areas are involved initial pain may be quite transient, where patient sinks immediately into shock. Spasm and perecordial distress quite often precedes severe prostration.

The initial shock is termed as primary shock in destinction from the later secondary or toxemic shock. One hundred years ago the sergeon termed this immediate shock a "consitutional burn". The blood pressure is low, pulse irregular, rapid soft, and very weak. The respiration becomes rapid, shallow and irregular. The temperature subnormal. The skin is cold and clammy.

At first, bowels are usually constipated, but this frequently is superseded by diarrhea, which exhausts the patient's strength with rapidity more than proportional to its apparent violence.

If the patient does not die during above period he enters a period of reaction and inflammation, which lasts from four to fourteen days. This reaction is of two types:

1. The sthenic type, in which patient suffers violence of inflammatory fever and,
2. The asthenic type in which the patient sinks under the febrile tumult at an early period.

The inflammatory reaction may be so serious as to imperil life; the internal organs are in a state of active congestion; there is general fever; the pulse is frequent and full; the skin parched and hot, and the tongue hot and dry.

The first shock is vasomotor in origin, the secondary shock is a toxanic shock due to inflammatory reaction. Cases that recover show subsidence of symptoms by the fifth or sixth day, while others continue to approach insiduously the state of toxemic shock. The symptoms become progressively more severe and hyperpnea heralds the approach of death in this toxemic shock.

Blood pressure is low, especially in systolic pressure, which is believed due to high concentration of blood and decrease of volume.

Temperature has a close relationship to blood volume. It is directly related to blood volume and concentration. The later, much higher temperature is

to be interpreted as a sequence of infection of injured area, or stimulation of medullary heat- Center by absorbed toxins; first degree burns do not effect body temperature.

After about ten days the slough separates from the burned area and healing begins. Suppuration frequently complicates this last stage.

The local symptoms are the following, in a first degree burn four common symptoms of imflammation are present:

1. Heat 2. Redness 3. Pain 4. Swelling

The redness may or may not be completely obliterated by pressure.

Burns of second degree have the following local symptoms, more painful than first degree, the pain being somewhat proportional to the tenseness of the vesicles. Burns of the third degree are the most painfull while deeper degrees are painless to the extent of nerve terminal destruction.

Burns involving the anal region, constipation is quite often the result, because pain experienced during defecation prohibits the patient to perform this act freely, if gential involved, dysuria may result.

Acid urine aggravates the above pathology. Diagnosis is usually very obvious. Until slough has separated it is very difficult, if not impossible, to pronounce with certainty whether a burn is of the third, fourth, or fifth degree.

The fourth degree is distinguished from the third by the fact that the eschar of the former is not sensitive to deep pressure at an early period; whereas, in the third degree, because of the survival and exposure of the sensitive nerve terminals, pressure produces the most intense agony. The fourth degree is distinguished from the fifth degree by the lack or absence of resonance or sonority on percussion of the eschar. The eschar of the fourth degree burn is thinner than that of the fifth degree, from which a distinctly sonorous percussion note can be elicited.

Careful examination is sometimes necessary to differentiate the varieties of burns. The distinction between burns and scalds at times occasions difficulty In scalds, the skin usually has an ashy hue, is never blackened or charred, is usually of a soft, pulpy, soaked or sodden appearance, is frequently covered with blisters, and the hairs usually persist and are not

scorched. In burns the skin is often charred, hard, leathery, dry and brown, and the hairs of the burned area are destroyed and those in the immediate vicinity scorched. Burns by petroleum or its derivatives resembles burns from flame, in that the skin near the burned area is not only scorched but blackened by a carbon deposit. The odor is noticeable and significant. Burns by mineral acids are easily distinguished as their eschar is soft and crumbling, is definitely demarcated, is not associated with blister formation, nor scorching of the hair and blackening of the surrounding skin.

Complication of burns are the following. The most common complication of burns and scalds is a secondary pyogenic infection of the burned area. The heat causing the burn sterilizes the skin and tissues, so that all lesions of this sort are primarily sterile. This is usually short lived because of the frequent contamination during the accident. The dead and devitalized tissues offer a most suitable soil. Practically every burn is infected within a few hours. The infection is a mixed type.

Suppuration begins, and is the result of three factors:

1. Cell necrosis
2. Local accumulation of leukocytes
3. Digestion of necrotic cells, fibrin, tissue elements by ferments derived from leukocytes.

This hastens sloughing. The sepsis is early and easily recognized by its peculiar and distinctly mephitic odor.

Septicemia is a dangerous sequel to burns, tetanus is more serious and common in burns of the head and trunk. Hemorrhage, menengites, apoplexy in elder individuals. Bed sores, bronchites, pneumonis, nephritis, duodenal ulcer, cicatrices and contractural deformites.

Prognosis of Burns and Scalds.

Mac Leod (31) believes prognosis of first and second degree burns are very good due to healing usually without scar tissue formation. Where the burn is deep in corium, more or less severe scarring results with contracture. The danger from burns is dependent on the surface area rather than depth of burn.

As far back as the year of 1919 Sherman (3) had some very good results in treatment of burns. Sherman had several cases where seventy-five to ninty per cent of surface body area was involved and patient recovered by the use of paraffin wax. More than fifty thousand cases where treated by this method. This treatment usually relieved pain in majority of cases after the first twenty-four to forty-eight hours.

Burns and scalds (4) always demand a guarded prognosis. In case of extensive burns the patient can never be reckoned safe until the whole has been fairly cicatrized. The prognosis varies with a series of influential factors which are:

1. The nature of burning agent
2. The age, sex, occupation and individual tolerance of patient.

3. The extent of the body surface affected.
4. The degree of depth of the burn involvement.
5. The particular region of the body burned.
6. The incidence of unusual and significant symptoms has a grave prognostic import.

The Causes of Death from Burns and Scalds: There are many theories as the possible cause of death due to burns as related by Park and Davis (4).

1. Respiratory function-leading to overwork in lungs or asphyxia. Yet the skin performs less than four per cent of the respiration of the body.
2. Others believe the protective funtions of normal skin is destroyed and death results from secondary bacteria.
3. Then, there are the theories that death is due to excitation or depression of the nervous system.
4. There are the theories that death is due to heart paralysis caused by absorption of toxin in blood.
5. Others believe death is due to alterations in circulative of blood.

Treatment

It is impossible to treat all burns alike. Each burned patient is an individual problem. (Park and Davis 4). The degree of burn is important and the surface area, the severity of symptomes and the condition and age of the patient.

The indication for the treatment of burns are:

To relieve pain and shock, guard against complication, especially of internal organs, promote healing, and return normal function and appearance.

A treatment must have the following qualifications:

1. Absolutely sterile.
2. Must be comfortable to the patient.
3. Must not adhere to wound because it would act as irritant.
4. Must be easily removed without causing pain
5. Should be non-toxic.
6. Should not interfere with epidermatization.
7. It should promote healing.

Degree of Shock: The very first observation of a severely burned patient is for degree of shock. An immediate hypodermic of morphine and atropine is indicated. The entire body should be rolled in a blanket and conveyed

to a hospital in a closed car.

Upon entering hospital if pain becomes severe, the hyperdermic should be repeated of morphine and atropine.

If patient is in shock, he has subnormal temperature an immediate application of heat is indicated. This case the system is treated and not the local injury.

When shock is overcome, and heat-regulating, mechanism received, the patient may be immersed in hot bath at temperature of 110° F. Here, under water the clothing may be removed, thus avoiding pain.

The local treatment may be so painful that patient might require nitrous oxide inhalation. This also tends to elevate the blood pressure. The burned area should be thoroughly cleaned, together with surrounding normal integument. Then, local treatment is started as best suited for the patient.

The one of greatest value is to prevent absorption of toxic from the injured tissue, tannic acid is the best for this, also picric acid. Intake and output of fluids is very important.

Heavy forced fluid in-take should continue until tendency of toxemia disappears and burned area becomes clean and granulating. A daily record should be kept

of in-take and out-put. Water by mouth, vein and glucose parentrally will convert urinary suppression to diuresis

Saline infussion intravenously not only dilutes and increase blood volume but also act as diuretic. Davidson observed that blood clorides remained at a low level as long as any sloughs are present. When all the burned, devitalized tissue separates and absorption is no longer taking place, the blood chlorides rise with a parallel rise of urinary chlorides.

Glucose solution is very useful in that it also furnishes the above with addition of furnishing nourishment. In severe toxic conditions dosage 1,000 to 2,000 CC of 5% glucose should be given slowly intravenously. It is ~~soft and~~ very helpfull to give fifteen units of insulm with above dosage of glucose.

Blood transfusion are very useful in restoring normal blood pressure.

Vaccines therapy: Mixed vaccines have been reported as useful in combating a frequent complication of burns. Such a vaccine may help some in prevention of bacteremia and septicemia.

Diet and General Measures:

Diet: In severe burns (4) the diet the first few days

should consist mostly of liquids. Hot coffee, beef tea, milk punch, given in frequent but small quantities. Diarrhea is best controlled by opium and bismuth. Constipation should of course, be treated with enemas.

General Measures: Fever approaching 103 F relief is best obtained by sponging with alcohol.

The management of minor burns of the first and second degree: The primary object of local treatment of erythematous burns is for pain and prevent effusion.

Cold water emmersion: Cold will very quickly remove free heat and prevent any further injury. Topical application of ice are analgetic during first three or four hours. The burned area may be immersed in water at a temperature of 55° F until the sensation of burning has been dulled, when the temperature of water is augmented to 70° F.

Antiseptic Lotion: Skepticism exists concerning the use of antiseptic solutions. ~~There is~~ On denuded areas ~~and by~~ adding an antiseptic acts as an irritant. For pain, light ointments, creams etc. are used and seem to help some. The best results are obtained by varnishing the surface with a thin layer of collodion, which acts as an insensible cuticle.

Care of Vesication: The serous contents of blisters, being in the first instance sterile, form a non-irritating dressing for the delicate underlying tissues. When blisters are left to rupture spontaneous the result is not as good as when drained under sterile conditions; therefore, sterile drainage at the most depended portion of the blister is indicated. Skin grafts from blister epithelium is not notably successful.

There are three reasons for use of local wet dressings:

1. To prevent toxemia
2. To control infection and
3. To hasten healing.

Park and Davis (5) give the following classification of treatment:

1. Solution which produce a local coagulation of devitalized tissues
    a. tannic acid
    b. picric acid
    c. alcohol
2. Solution which is active in arresting the autolytic process in burned tissue

a. Sodium Bicarbonate

3. Solution which slows the process of toxic absorption by the local use of vaso-constrictor drugs.

A. Adrenalin.

4. Solutions which effect chemical debridement by removing the products of tissue decomposition.

a. Acetic acid

b. Dakin's solution

5. Hypertonic solutions:

a. Twice normal saline solution

b. Concentrated magnesium sulphate solution.

6. Solutions for the treatment of wound infection.

a. The chlorine antiseptics

b. Mercurochrome

c. Neutral acriflavine

7. Solutions whose usage is quetionable, futile or dangerous.

a. Normal saline solution.

b. Boric acid.

c. Aluminium acetate

d. Potassium permanganate.

e. Mercuric chloride

f. Carbolic acid.

g. Turpentine.

h. Iodine.

i. Chloretone.

Method of Application of Tannic Acid.

It is of interest to know the physical and chemical property of this drug. It is a non-nitrogenous amorphous powder, which is readily soluble in water and alcohol, and forms a more or less stable compound with protein constituents of body fluid and cells.

On a burned surface it acts as an astringent effect on the superfical tissue. It serves the following ways, protection against, chemical, bacterial, and mechanical action as well as against sensory and inflammatory irritation.

Method of Application.

After everything is under control one turns to treat the local wound. After this has been cleaned as stated before, the area is ready for tannic acid.

The areas are first covered with sterile dry gauze. This is then soaked with 2.5% aquaeous solution of tannic acid. It is important to have fresh preparation mixed because in standing it loses its action.

In order to prevent deep caustic reaction from the tannic acid, small sections of dressing are removed at the end of 18-24 hours. As soon as all red areas have subsided, and have obtained a light brown color, all dressings are removed.

If the dressing has dried to the wound it should be first soaked with tannic acid solution before further removing. The wound is then left exposed to air. One must be careful to protect the area from bacterial invasion and chilling.

Treatment of Face

A five per cent tannic acid ointment has been used by Davidson around eyes with safety.

Use of Tannic Acid Spray

Tannic Acid may be sprayed on burned surfaces with an atomizer. The wound is covered with a five% spray every half hour until the surface becomes mahogany brown.

Extensive burns require a coagulated period of sixteen hours. Exposure to air hastens the coagulation process. The tannic acid crust usually separates

between the fourteenth and twentieth day leaving a clear granulating surface to facilitate the removal of these crusts; oil is preferiable to acquaeous solution, because the latter favors the latent absorption of previously imprisoned burn toxins.

If pain developes over area with patient having fever, one must induce free drainage and tannic crust must be widely removed and burn treated by careful application of anteseptic solutions.

Barnes (15) gives the following results. In a series of two hundred and five burns, two of every three deaths took place in the first twenty-four hours. Therefore, the primary treatment in saving the life and preservation of function being second.

It is interesting to see that Davidson originally used 2.5% tannic acid solution (Barnes 15) because of concentration might result in caustic acid. Barnes reports using 10% tannic acid in order to penetrate to the bottom of the burn and to secure at once a sterile scab. Barnes has no fear that stronger solution will damage living tissue. Deep penetration seals the capillary bed and by conservation of body fluids prevents concentration of the blood.

(Wilson 32). The prognosis is much better since the use of tannic acid espicially in children. Twelve years ago one-eight of burned surface area was almost always certain death. With present methods of treatment one-third body surface involved now causes anxiety only in exceptional circumstances.

(Taylor 33) following is the summary of misuse of tannic acid treatment.

1. The tannic acid treatment of burns was originally advocated for use in severe, extreme cases. It is now frequently used in milder, "second degree" burns.

2. The action of tannic acid applied to a burned area in which viable island of the germinal epitheral cells still survive is not limited to the dead tissue. Many of the epitheral cells that might take part in the repair of the denuded area are also tanned by the treatment. Repair is thus delayed.

3. It is suggested that the coagulation treatment of burns be reserved for the most severe types and the bland wet dressing and ointment be used on the great majority of "second degree" burns.

(Camp 30) Tannic acid is contra-indicated in wounds probably infected and is not effective where

used with oily ointments. These ointments have proven themselves to be one of the poorest types of burn dressing and it is well to urge that they be not used. If ointment has been previously applied it may be in many cases be removed with zylene without great pain.

Picric Acid

According to Davis and Parks(4) picric acid should be limited to emergency treatment of small burns and scalds.

It is used as 1.2 per cent strength in aquaeous solution. When applying, if blister present they should be opened so that the solution may cover these areas. It has been shown this drug is toxic and, therefore, not safe to use over large areas.

Alger contends that the combination of picric acid and citric acids, which Esbach devised for the detection of albumen is more effective than picric acid alone, in burns of the second degree. Esbachs solution consists of ten parts of picric acid, twenty parts of citric acid and one thousand part water.

It is believed that the picric acid acts in the following manner. Over any denuded surface it forms

a protective, aseptic scab, by coagulation of secreted serum, which heals up ruptured lymph spaces, protects exposed nerve endings, and splints the wound in such a fashion that epilheleal profiferation may proceed rapidly beneath, simulating nature's method. The artificial scab as in tannic acid treatment promotes rapid healing prevents external infection and reduces pain.

Acetic Acid Treatment

Park and Davis (4) have found the following results 0.5% to 2% sterile acetic acid was used in which Turkish towels were soaked and applied direct to area involved. Acetic acid treatment acts opposite of what the other treatment acts. It digests the disturbed tissue area and decreases the time between skin grafting and does prevent early contractures.

Use of Antiseptics

When deudation occurs and measures for abortion of infection namely, tanning, drying, dibridement, etc, have failed; then the surgeon must have recourse to the gredicious and cautious use of antiseptic solution which usually cause more harm than good. No phycician

should employ the constant application of antiseptic solutions to burned areas as a means to prevent infection, because other methods are so superior and lack the disadvantage of antisepsis. (Greenwold 34).

Summary

The present up to date therapy of burns is far from being ideal. There is no question of the great improvement within the last five years, resulting in a much more favorable prognosis.

Six or seven years ago, a burned surface area covering one eighth of body surface, most always was fatal. With present day knowledge and therapy a burned surface area covering one third of body surface carries a good prognosis. There are burn cases now reported in which 70 to 90 per cent of body surface is involved, with the saving of those lifes.

The Classification of burns as to "degree" is of little clinical importance. The greatest factor is the extent of surface area involved.

The greatest asset in therapy is the proper first aid treatment; the use of coagualable drugs on burned areas and the intravenous colloid solution.

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